```
     FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002
L1        19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
L2          509 S PROLYL ?HYDROXYLASE
L3            2 S L1 (S) L2
L4            2 S L1 (L) L2
L5            7 S DPY-18
L6          103 S DPY-18 OR DPY
L7           59 S L1 (L) L6
L8            0 S L7 AND L2

=> FILE CAPLUS MEDLINE BIOSIS EMBASE
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       3.06       3.27

FILE 'CAPLUS' ENTERED AT 14:49:21 ON 18 JUL 2002
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FILE 'MEDLINE' ENTERED AT 14:49:21 ON 18 JUL 2002

FILE 'BIOSIS' ENTERED AT 14:49:21 ON 18 JUL 2002
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FILE 'EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002
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=> S L3
L9           11 L3

=> DUP REM L9
PROCESSING COMPLETED FOR L9
L10           6 DUP REM L9 (5 DUPLICATES REMOVED)

=> D 1-6 IBIB ABS

L10  ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2002 ACS         DUPLICATE 1
ACCESSION NUMBER:         2001:757463  CAPLUS
DOCUMENT NUMBER:          136:33452
TITLE:                    C. elegans EGL-9 and mammalian homologs define a
                          family of dioxygenases that regulate HIF by prolyl
                          hydroxylation
AUTHOR(S):                Epstein, Andrew C. R.; Gleadle, Jonathan M.; McNeill,
                          Luke A.; Hewitson, Kirsty S.; O'Rourke, John; Mole,
                          David R.; Mukherji, Mridul; Metzen, Eric; Wilson,
                          Michael I.; Dhanda, Anu; Tian, Ya-Min; Masson, Norma;
                          Hamilton, Donald L.; Jaakkola, Panu; Barstead, Robert;
                          Hodgkin, Jonathan; Maxwell, Patrick H.; Pugh,
                          Christopher W.; Schofield, Christopher J.; Ratcliffe,
                          Peter J.
CORPORATE SOURCE:         The Henry Wellcome Building of Genomic Medicine,
                          Oxford, OX3 7BN, UK
SOURCE:                   Cell (Cambridge, MA, United States) (2001), 107(1),
                          43-54
                          CODEN: CELLB5; ISSN: 0092-8674
PUBLISHER:                Cell Press
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   HIF is a transcriptional complex that plays a central role in mammalian
     oxygen homeostasis.  Recent studies have defined posttranslational
     modification by prolyl hydroxylation as a key regulatory event that
     targets HIF-.alpha. subunits for proteasomal destruction via the von
     Hippel-Lindau ubiquitylation complex.  Here, we define a conserved
     HIF-VHL-  ***prolyl***     ***hydroxylase***   pathway in   ***C***  .
       ***elegans***  , and use a genetic approach to identify EGL-9 as a
     dioxygenase that regulates HIF by prolyl hydroxylation.  In mammalian
     cells, we show that the HIF-prolyl hydroxylases are represented by a
     series of isoforms bearing a conserved 2-histidine-1-carboxylate iron
     coordination motif at the catalytic site.  Direct modulation of
     recombinant enzyme activity by graded hypoxia, iron chelation, and
     cobaltous ions mirrors the characteristics of HIF induction in vivo,
     fulfilling requirements for these enzymes being oxygen sensors that
     regulate HIF.
REFERENCE COUNT:          40    THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
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L10 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:324546 CAPLUS
DOCUMENT NUMBER: 133:86929
TITLE: Prolyl 4-hydroxylase is an essential procollagen-modifying enzyme required for exoskeleton formation and the maintenance of body shape in the nematode Caenorhabditis elegans
AUTHOR(S): Winter, Alan D.; Page, Antony P.
CORPORATE SOURCE: Wellcome Centre for Molecular Parasitology, Anderson College, The University of Glasgow, Glasgow, G11 6NU, UK
SOURCE: Molecular and Cellular Biology (2000), 20(11), 4084-4093
CODEN: MCEBD4; ISSN: 0270-7306
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The multienzyme complex prolyl 4-hydroxylase catalyzes the hydroxylation of proline residues and acts as a chaperone during collagen synthesis in multicellular organisms. The .beta. subunit of this complex is identical to protein disulfide isomerase (PDI). The free-living nematode C. elegans is encased in a collagenous exoskeleton and represents an excellent model for the study of collagen biosynthesis and extracellular matrix formation. In this study, we examd. prolyl 4-hydroxylase .alpha.-subunit (PHY; EC 1.14.11.2)- and .beta.-subunit (PDI; EC 5.3.4.1)-encoding genes with respect to their role in collagen modification and formation of the C. elegans exoskeleton. We identified genes encoding 2 PHYs and a single assocd. PDI and showed that all 3 are expressed in collagen-synthesizing ectodermal cells at times of maximal collagen synthesis. Disruption of the pdi gene via RNA interference resulted in embryonic lethality. Similarly, the combined phy genes are required for embryonic development. Interference with phy-1 resulted in a morphol. dumpy phenotype, which we detd. to be identical to the uncharacterized dpy-18 locus. Two dpy-18 mutant strains were shown to have null alleles for phy-1 and to have a reduced hydroxyproline content in their exoskeleton collagens. This study demonstrates in vivo that this enzyme complex plays a central role in extracellular matrix formation and is essential for normal metazoan development.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 6 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 79021663 MEDLINE
DOCUMENT NUMBER: 79021663 PubMed ID: 212107
TITLE: In vitro translation of nematode cuticular collagens.
AUTHOR: Noble S; Leushner J; Pasternak J
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1978 Aug 23) 520 (1) 219-28.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197812
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19781220

AB Phenanthroline treatment of growing cultures of the free-living ***nematode*** Panagrellus silusiae was used to lower the degree of hydroxylation of nascent collagen chains at the polysomal level. Under these conditions, the bound pentasome-hexasome fraction provided substrate for ***prolyl*** ***hydroxylase*** . When this polysomal fraction was subsequently tested in a cell-free wheat germ system, collagenase-susceptible translation products were observed after sodium dodecyl sulfate-acrylamide gel electrophoresis. The electrophoretic mobilities of each of these four major collagen products were similar to four collagens that are isolated from intact cuticles. In addition, purified polysomal RNA that adhered to unmodified cellulose directed the synthesis of four pepsin-resistant polypeptides that had molecular weights that coincided with four pepsin-resistant collagens that can be purified from the cuticle of this species. Thus, the polysomal site of the messenger RNAs for the cuticular collagens of P. silusiae was located. Although precursor forms of the cuticular collagens were not produced in the cell-free system, the question whether additional amino acid segments occur on the primary translational products of the cuticular collagens in vivo remains open.

L10 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
ACCESSION NUMBER: 1978:185080 CAPLUS
DOCUMENT NUMBER: 88:185080
TITLE: Partial purification and characterization of ***prolyl*** ***hydroxylase*** from the free-living ***nematode*** Panagrellus silusiae
AUTHOR(S): Leushner, J. R. A.; Pasternak, J.
CORPORATE SOURCE: Dep. Biol., Univ. Waterloo, Waterloo, Ont., Can.
SOURCE: Can. J. Zool. (1978), 56(2), 159-65
CODEN: CJZOAG; ISSN: 0008-4301
DOCUMENT TYPE: Journal
LANGUAGE: English
AB ***Prolyl*** ***hydroxylase*** (I) was partially purified from the ***nematode*** P. silusiae and its physicochem. and biol. properties were studied. I purifn. involved (NH4)2SO4 pptn. and Ca phosphate gel ion exchange from Triton X-100-treated Panagrellus homogenates. Gel filtration indicated a mol. wt. of .apprx.285,000; acrylamide electrophoresis showed the component to be comprised of subunits having mol. wts. of .apprx.67,000. I activity was dependent on .alpha.-ketoglutarate, Fe2+, ascorbate, catalase, O, and dithiothreitol. Activity was inhibited by .alpha.,.alpha.-dipyridyl, phenanthroline, and polyproline. The Km value for the substrate was 80 .mu.g.

L10 ANSWER 5 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1978:67206 BIOSIS
DOCUMENT NUMBER: BR15:10706
TITLE: PROTO COLLAGEN ***PROLYL*** ***HYDROXYLASE*** IN THE FREE LIVING ***NEMATODE*** PANAGRELLUS-SILUSIAE.
AUTHOR(S): LEUSHNER J R A; PASTERNAK J J
SOURCE: Proc. Can. Fed. Biol. Soc., (1976) 19, 98.
CODEN: PCBSA2.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: Unavailable

L10 ANSWER 6 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 76171264 EMBASE
DOCUMENT NUMBER: 1976171264
TITLE: Programmed synthesis of collagen during postembryonic development of the nematode Panagrellus silusiae.
AUTHOR: Leushner J.; Pasternak J.
CORPORATE SOURCE: Dept. Biol., Univ. Waterloo, Canada
SOURCE: Developmental Biology, (1975) 47/1 (68-80).
CODEN: DEBIAO
DOCUMENT TYPE: Journal
FILE SEGMENT: 029 Clinical Biochemistry
021 Developmental Biology and Teratology
LANGUAGE: English
AB The relative rate of collagen synthesis in the free living ***nematode*** Panagrellus silusiae during postembryonic development was found to be discontinuous by measuring either the incorporation of tritium into material extracted as collagen or the amount of collagen bound tritiated proline and hydroxyproline after 2 hr incubations of whole worms with [3H]proline. A peak of collagen production preceded each of the three molts that were examined. Moreover, protocollagen ***prolyl*** ***hydroxylase*** activity during each intermolt period paralleled the pattern of collagen synthesis. On the other hand, a triphasic pattern was not observed when noncollagenous proteins were labeled with either [3H]tryptophan or [3H]leucine. In addition, the level of soluble radioactive proline that accumulates in whole organisms after 2 hr incubation periods did not fluctuate appreciably during postembryonic development. The mean ratio of hydroxyproline to proline in a number of collagen samples extracted at various times during the maturation phase was 0.113 .+-. 0.040. Pulse and chase experiments with [3H]proline indicated that most of the collagen synthesized during a peak period is lost after the second ecdysis following the labeling interval. In contrast, a considerable proportion of the collagen synthesized during nonpeak periods is retained throughout the postembryonic period. It is postulated that the modulated pattern of collagen biosynthesis in Panagrellus reflects, for the most part, a quantitative regulation of the production of cuticular collagen during postembryonic development.

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

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     FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002
L1         19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
L2           509 S PROLYL ?HYDROXYLASE
L3             2 S L1 (S) L2
L4             2 S L1 (L) L2
L5             7 S DPY-18
L6           103 S DPY-18 OR DPY
L7            59 S L1 (L) L6
L8             0 S L7 AND L2

     FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002
L9            11 S L3
L10            6 DUP REM L9 (5 DUPLICATES REMOVED)

=> S L5
L11           25 L5

=> S EMBRYONAL LETHAL PHENOTYPE
L12            0 EMBRYONAL LETHAL PHENOTYPE

=> S EMBRYONIC LETHAL PHENOTYPE
L13          283 EMBRYONIC LETHAL PHENOTYPE

=> S EMBRYON? LETHAL PHENOTYPE
L14          290 EMBRYON? LETHAL PHENOTYPE

=> S EMBRYON? LETHA? PHENOTYPE
L15          290 EMBRYON? LETHA? PHENOTYPE

=> S L1 (s) L15
L16           35 L1 (S) L15

=> S L16 AND L2
L17            0 L16 AND L2

=> S L1 (s) L15  (s) L2
L18            0 L1 (S) L15 (S) L2

=> S L1 (L) L15  (L) L2
L19            0 L1 (L) L15 (L) L2

=> D HIS

     (FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

     FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002
L1         19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
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L4             2 S L1 (L) L2
L5             7 S DPY-18
L6           103 S DPY-18 OR DPY
L7            59 S L1 (L) L6
L8             0 S L7 AND L2

     FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002
L9            11 S L3
L10            6 DUP REM L9 (5 DUPLICATES REMOVED)
L11           25 S L5
L12            0 S EMBRYONAL LETHAL PHENOTYPE
L13          283 S EMBRYONIC LETHAL PHENOTYPE
L14          290 S EMBRYON? LETHAL PHENOTYPE
L15          290 S EMBRYON? LETHA? PHENOTYPE
L16           35 S L1 (S) L15
L17            0 S L16 AND L2
L18            0 S L1 (S) L15  (S) L2
L19            0 S L1 (L) L15  (L) L2

=> DUP REM L16
PROCESSING COMPLETED FOR L16
L20           12 DUP REM L16 (23 DUPLICATES REMOVED)

=> S L6
L21          421 L6

=> S L21 (s) L1
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=> S L21 AND L22
L23         222 L21 AND L22

=> S L1 (s) L15 (s) L21
L24           0 L1 (S) L15 (S) L21

=> S L1 (L) L15 (L) L21
L25           0 L1 (L) L15 (L) L21

=> S L1 AND L15 AND L21
L26           1 L1 AND L15 AND L21

=> D

L26  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2002 ACS
AN   1998:454514  CAPLUS
DN   129:212270
TI   Isolation and characterization of lethal mutation near the unc-29 (LG I)
     region of Caenorhabditis   ***elegans***
AU   Lee, Jinsook; Ahnn, Joohong
CS   Department of Life Science, Kwangju Institute of Science and Technology,
     Kwangju, 506-712, S. Korea
SO   Korean Journal of Biological Sciences (1998), 2(1), 123-131
     CODEN: KJBSFZ; ISSN: 1226-5071
PB   Korean Association of Biological Sciences
DT   Journal
LA   English


=> D IBIB ABS

L26  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         1998:454514  CAPLUS
DOCUMENT NUMBER:          129:212270
TITLE:                    Isolation and characterization of lethal mutation near
                          the unc-29 (LG I) region of Caenorhabditis
                            ***elegans***
AUTHOR(S):                Lee, Jinsook; Ahnn, Joohong
CORPORATE SOURCE:         Department of Life Science, Kwangju Institute of
                          Science and Technology, Kwangju, 506-712, S. Korea
SOURCE:                   Korean Journal of Biological Sciences (1998), 2(1),
                          123-131
                          CODEN: KJBSFZ; ISSN: 1226-5071
PUBLISHER:                Korean Association of Biological Sciences
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   The unc-29 region on the chromosome I of Caenorhabditis   ***elegans***
     has been mutagenized in order to obtain lethal mutations.  In this screen,
     the uncoordinated phenotype of unc-29 (e193) mutant was used to identify
     any lethal mutations closely linked to the unc-29 gene, which encodes a
     subunit of nicotinic acetylcholine receptors.  The authors have isolated
     six independent mutations (jh1 to jh6) out of approx. 5,200 Et
     methanesulfonate (EMS) treated haploids.  Four of the six mutations
     demonstrated   ***embryonic***    ***lethal***    ***phenotypes***  ,
     while the other two showed embryonic and larval lethal phenotypes.
     Terminal phenotypes obsd. in two mutations (jh1 and jh2) indicated
     developmental defects specific to posterior part of embryos which appeared
     similar to the phenotypes obsd. in nob (no back end) mutants.  Another
     mutation (jh4) resulted in an interesting phenotype of body-wall muscle
     degeneration at larval stage.  These mutations were mapped by using
     three-factor crosses and deficiency mutants in this region.  Here the
     authors report genetic anal. and characterization of these lethal
     mutations.


=> D HIS

     (FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

     FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002
L1         19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
L2           509 S PROLYL ?HYDROXYLASE
L3             2 S L1 (S) L2
L4             2 S L1 (L) L2
L5             7 S DPY-18
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L7            59 S L1 (L) L6
L8             0 S L7 AND L2

     FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002
L9            11 S L3
L10            6 DUP REM L9 (5 DUPLICATES REMOVED)
L11           25 S L5
L12            0 S EMBRYONAL LETHAL PHENOTYPE
L13          283 S EMBRYONIC LETHAL PHENOTYPE
L14          290 S EMBRYON? LETHAL PHENOTYPE
L15          290 S EMBRYON? LETHA? PHENOTYPE
L16           35 S L1 (S) L15
L17            0 S L16 AND L2
L18            0 S L1 (S) L15  (S) L2
L19            0 S L1 (L) L15  (L) L2
L20           12 DUP REM L16 (23 DUPLICATES REMOVED)
L21          421 S L6
L22          222 S L21 (S) L1
L23          222 S L21 AND L22
L24            0 S L1 (S) L15 (S) L21
L25            0 S L1 (L) L15 (L) L21
L26            1 S L1 AND L15 AND L21

=> DUP REM L22
PROCESSING COMPLETED FOR L22
L27           99 DUP REM L22 (123 DUPLICATES REMOVED)

=> S L1 AND L11
L28           21 L1 AND L11

=> S L27 AND L28
L29            8 L27 AND L28

=> DUP REM L29
PROCESSING COMPLETED FOR L29
L30            8 DUP REM L29 (0 DUPLICATES REMOVED)

=> D 1-8 IBIB ABS

L30  ANSWER 1 OF 8     MEDLINE
ACCESSION NUMBER:   2002354365     IN-PROCESS
DOCUMENT NUMBER:    22092148   PubMed ID: 12097347
TITLE:              High-Throughput Gene Mapping in Caenorhabditis
                      ***elegans***  .
AUTHOR:             Swan Kathryn A; Curtis Damian E; McKusick Kathleen B;
                    Voinov Alexander V; Mapa Felipa A; Cancilla Michael R
CORPORATE SOURCE:   Exelixis, Inc., South San Francisco, California 94083-0511,
                    USA.
SOURCE:             GENOME RESEARCH, (2002 Jul) 12 (7) 1100-5.
                    Journal code: 9518021. ISSN: 1088-9051.
PUB. COUNTRY:       United States
                    Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE:         Entered STN: 20020707
                    Last Updated on STN: 20020707
AB   Positional cloning of mutations in model genetic systems is a powerful
     method for the identification of targets of medical and agricultural
     importance. To facilitate the high-throughput mapping of mutations in
     Caenorhabditis   ***elegans***  , we have identified a further 9602
     putative new single nucleotide polymorphisms (SNPs) between two   ***C***
     .   ***elegans***   strains, Bristol N2 and the Hawaiian mapping strain
     CB4856, by sequencing inserts from a CB4856 genomic DNA library and using
     an informatics pipeline to compare sequences with the canonical N2 genomic
     sequence. When combined with data from other laboratories, our marker set
     of 17,189 SNPs provides even coverage of the complete worm genome. To
     date, we have confirmed >1099 evenly spaced SNPs (one every 91 +/- 56 kb)
     across the six chromosomes and validated the utility of our SNP marker set
     and new fluorescence polarization-based genotyping methods for systematic
     and high-throughput identification of genes in   ***C***  .
       ***elegans***   by cloning several proprietary genes. We illustrate our
     approach by recombination mapping and confirmation of the mutation in the
     cloned gene,   ***dpy***  -  ***18***  . [The sequence data described in
     this paper have been submitted to the NCBI dbSNP data library under
     accession nos. 4388625-4389689 and GenBank dbSTS under accession nos.
     973810-974874. The following individuals and institutions kindly provided
```

The ***C*** . ***elegans*** Sequencing Consortium and The Caenorhabditis Genetics Center.]

L30 ANSWER 2 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:141383 BIOSIS
DOCUMENT NUMBER: PREV200200141383
TITLE: The T-box factor MLS-1 acts as a molecular switch during specification of nonstriated muscle in ***C*** . ***elegans*** .
AUTHOR(S): Kostas, Stephen A.; Fire, Andrew (1)
CORPORATE SOURCE: (1) Department of Embryology, Carnegie Institution of Washington, Baltimore, MD, 21210: fire@ciwemb.edu USA
SOURCE: Genes & Development, (January 15, 2002) Vol. 16, No. 2, pp. 257-269. http://www.genesdev.org/. print.
ISSN: 0890-9369.
DOCUMENT TYPE: Article
LANGUAGE: English
AB We have isolated mutations in a gene mls-1 that is required for proper specification of nonstriated muscle fates in Caenorhabditis ***elegans*** . Loss of MLS-1 activity causes uterine muscle precursors to forego their normal fates, instead differentiating as vulval muscles. We have cloned mls-1 and shown that the product is a member of the T-box family of transcriptional regulators. MLS-1 acts as a cell fate determinant in that ectopic expression can transform other cell types to uterine muscle precursors. Uterine muscle patterning is executed by regulation of MLS-1 at several different levels. The mls-1 promoter is activated by the ***C*** . ***elegans*** orthologs of Twist and Daughterless, but is only active in a subset of the lineage where these two transcription factors are present. mls-1 activity also appears to be regulated by posttranscriptional processes, as expression occurs in both uterine and vulval muscle precursors.

L30 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:68425 BIOSIS
DOCUMENT NUMBER: PREV200200068425
TITLE: Role of ***C*** . ***elegans*** lin-40 MTA in vulval fate specification and morphogenesis.
AUTHOR(S): Chen, Zhe; Han, Min (1)
CORPORATE SOURCE: (1) Department of Molecular, Cellular and Developmental Biology, Howard Hughes Medical Institute, University of Colorado at Boulder, Boulder, CO, 80309: mhan@colorado.edu USA
SOURCE: Development (Cambridge), (December, 2001) Vol. 128, No. 23, pp. 4911-4921. http://dev.biologists.org/current.shtml. print.
ISSN: 0950-1991.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Vulval differentiation in Caenorhabditis ***elegans*** involves several fundamental cellular events, including cell fusion, division and migration. We have characterized the role of the lin-40 (also known as egr-1) gene in these cellular processes. LIN-40 is homologous to the metastasis-associated factor 1 (MTA1) in mammals, which has been identified as a component of the nucleosome remodeling and histone deacetylation (NuRD) complex that functions as a transcriptional co-repressor. We show here that lin-40 negatively regulates vulval fate specification at least partly by promoting cell fusion between the vulval precursor cells and the hypodermal syncytium at an early larval stage. This inhibitory function of lin-40 might be carried out by downregulating lin-39 Hox expression. We also show that lin-40 is specifically required for cell divisions along the transverse orientation during vulval morphogenesis.

L30 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:296070 CAPLUS
DOCUMENT NUMBER: 133:233493
TITLE: Prolyl 4-hydroxylase is required for viability and morphogenesis in Caenorhabditis ***elegans***
AUTHOR(S): Friedman, Lisa; Higgin, Joshua J.; Moulder, Gary; Barstead, Robert; Raines, Ronald T.; Kimble, Judith
CORPORATE SOURCE: Department of Biochemistry, University of Wisconsin, Madison, WI, 53706, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2000), 97(9), 4736-4741
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences

LANGUAGE: English
AB The genome of Caenorhabditis ***elegans*** possesses two genes, ***dpy*** - ***18*** and phy-2, that encode .alpha. subunits of the enzyme prolyl 4-hydroxylase. Th authors have generated deletions within each gene to eliminate prolyl 4-hydroxylase activity from the animal. The ***dpy*** - ***18*** mutant has an aberrant body morphol., consistent with a role of prolyl 4-hydroxylase in formation of the body cuticle. The phy-2 mutant is phenotypically wild type. However, the ***dpy*** - ***18*** ; phy-2 double mutant is not viable, suggesting an essential role for prolyl 4-hydroxylase that is normally accomplished by either ***dpy*** - ***18*** or phy-2. The effects of the double mutation were mimicked by small-mol. inhibitors of prolyl 4-hydroxylase, validating the genetic results and suggesting that ***C*** . ***elegans*** can serve as a model system for the discovery of new inhibitors.
REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:324546 CAPLUS
DOCUMENT NUMBER: 133:86929
TITLE: Prolyl 4-hydroxylase is an essential procollagen-modifying enzyme required for exoskeleton formation and the maintenance of body shape in the ***nematode*** Caenorhabditis ***elegans***
AUTHOR(S): Winter, Alan D.; Page, Antony P.
CORPORATE SOURCE: Wellcome Centre for Molecular Parasitology, Anderson College, The University of Glasgow, Glasgow, G11 6NU, UK
SOURCE: Molecular and Cellular Biology (2000), 20(11), 4084-4093
CODEN: MCEBD4; ISSN: 0270-7306
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The multienzyme complex prolyl 4-hydroxylase catalyzes the hydroxylation of proline residues and acts as a chaperone during collagen synthesis in multicellular organisms. The .beta. subunit of this complex is identical to protein disulfide isomerase (PDI). The free-living ***nematode*** ***C*** . ***elegans*** is encased in a collagenous exoskeleton and represents an excellent model for the study of collagen biosynthesis and extracellular matrix formation. In this study, we examd. prolyl 4-hydroxylase .alpha.-subunit (PHY; EC 1.14.11.2)- and .beta.-subunit (PDI; EC 5.3.4.1)-encoding genes with respect to their role in collagen modification and formation of the ***C*** . ***elegans*** exoskeleton. We identified genes encoding 2 PHYs and a single assocd. PDI and showed that all 3 are expressed in collagen-synthesizing ectodermal cells at times of maximal collagen synthesis. Disruption of the pdi gene via RNA interference resulted in embryonic lethality. Similarly, the combined phy genes are required for embryonic development. Interference with phy-1 resulted in a morphol. dumpy phenotype, which we detd. to be identical to the uncharacterized ***dpy*** - ***18*** locus. Two ***dpy*** - ***18*** mutant strains were shown to have null alleles for phy-1 and to have a reduced hydroxyproline content in their exoskeleton collagens. This study demonstrates in vivo that this enzyme complex plays a central role in extracellular matrix formation and is essential for normal metazoan development.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:550782 CAPLUS
DOCUMENT NUMBER: 134:247771
TITLE: ***dpy*** - ***18*** encodes an .alpha.-subunit of prolyl-4-hydroxylase in Caenorhabditis ***elegans***
AUTHOR(S): Hill, Katherine L.; Harfe, Brian D.; Dobbins, Carey A.; L'Hernault, Steven W.
CORPORATE SOURCE: Program in Genetics and Molecular Biology, Graduate Division of Biological and Biomedical Sciences, Emory University, Atlanta, GA, 30322, USA
SOURCE: Genetics (2000), 155(3), 1139-1148
CODEN: GENTAE; ISSN: 0016-6731
PUBLISHER: Genetics Society of America
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Collagen is an extracellular matrix (ECM) component encoded by a large

post-translationally modified by prolyl-4-hydroxylase (EC 1.14.11.2) before secretion and participation in ECM formation. Therefore, collagen processing and regulation can be studied by examg. this required interaction of prolyl-4-hydroxylase with procollagen. High-resoln. polymorphism mapping was used to place the Caenorhabditis ***elegans*** ***dpy*** - ***18*** gene on the phys. map, and we show that it encodes a prolyl-4-hydroxylase .alpha. catalytic subunit. The Dpy phenotype of ***dpy*** - ***18*** (e364) amber mutants is more severe when this mutation is in trans to the noncomplementing deficiency tDf7, while the ***dpy*** - ***18*** (e499) deletion mutant exhibits the same phenotype as ***dpy*** - ***18*** (e499)/tDf7. Furthermore, ***dpy*** - ***18*** RNA interference (RNAi) in wild-type worms results in Dpy progeny, while ***dpy*** - ***18*** (RNAi) in ***dpy*** - ***18*** (e499) mutants does not alter the Dpy phenotype of their progeny. These observations suggest that the ***dpy*** - ***18*** null phenotype is Dy. A ***dpy*** - ***18*** ::gfp promoter fusion construct is expressed throughout the hypodermis within the cells that abundantly produce the cuticle collagens, as well as in certain head and posterior neurons. While prolyl-4-hydroxylase has been studied extensively by biochem. techniques, this is the first report of a mutationally defined prolyl-4-hydroxylase in any animal.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:323602 BIOSIS
DOCUMENT NUMBER: PREV200000323602
TITLE: Mutations with sensory ray defect unmask cuticular glycoprotein antigens in Caenorhabditis ***elegans*** male tail.
AUTHOR(S): Ko, Frankie C. F.; Chow, King L. (1)
CORPORATE SOURCE: (1) Department of Biology, Hong Kong University of Science and Technology, Clear Water Bay, Kowloon, Hong Kong China
SOURCE: Development Growth & Differentiation, (Feb., 2000) Vol. 42, No. 1, pp. 69-77. print.
ISSN: 0012-1592.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Caenorhabditis ***elegans*** male tail has nine pairs of bilaterally symmetric ray processes extended into a cuticular fan. The formation of these structures involves both cell lineage differentiation and cellular morphogenesis. Nine mutations were examined, all of which presented an amorphous ray phenotype. Glycoconjugates carrying an N-acetylglucosamine (GlcNAc) epitope were detected at a high level in their male bursa. It was shown that these antigens are not responsible for the morphological defects. It was further demonstrated that these ram and mab gene products represent critical components for male tail cuticle organization. Mutations of them abolish the integrity of the male bursal cuticle and unmask the underlying GlcNAc epitope.

L30 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:116067 CAPLUS
DOCUMENT NUMBER: 114:116067
TITLE: Properties of a class of genes required for ray morphogenesis in Caenorhabditis ***elegans***
AUTHOR(S): Baird, Scott E.; Emmons, Scott W.
CORPORATE SOURCE: Dep. Mol. Genet., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SOURCE: Genetics (1990), 126(2), 335-44
CODEN: GENTAE; ISSN: 0016-6731
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Eight mutations were identified in Caenorhabditis ***elegans*** that define at least 5 terminal differentiation genes (ram genes) whose products are required during the extension of the male-specific ray sensilla. ram Gene mutations result in morphol. abnormalities in the sensory rays but do not appear to interfere with ray functions. A similar ray morphol. phenotype was obsd. in males harboring mutations in 3 previously defined genes, dpy-11, ***dpy*** - ***18*** , and sqt-1, that also affect body shape. One of these genes, sqt-1, is known to encode a collagen. Mutations in different ram genes failed to complement, suggesting that their gene products functionally interact. For one ram gene, failure to complement was shown to result from haploinsufficiency. Intergenic noncomplementation did not extend to the body morphol. genes. The temp.-sensitive periods of both ram and body morphol. mutations

It is proposed that ram gene products act together in a crit. interaction between the rays and the cuticle required for wild-type ray morphol.

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(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002
L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
L2 509 S PROLYL ?HYDROXYLASE
L3 2 S L1 (S) L2
L4 2 S L1 (L) L2
L5 7 S DPY-18
L6 103 S DPY-18 OR DPY
L7 59 S L1 (L) L6
L8 0 S L7 AND L2

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002
L9 11 S L3
L10 6 DUP REM L9 (5 DUPLICATES REMOVED)
L11 25 S L5
L12 0 S EMBRYONAL LETHAL PHENOTYPE
L13 283 S EMBRYONIC LETHAL PHENOTYPE
L14 290 S EMBRYON? LETHAL PHENOTYPE
L15 290 S EMBRYON? LETHA? PHENOTYPE
L16 35 S L1 (S) L15
L17 0 S L16 AND L2
L18 0 S L1 (S) L15 (S) L2
L19 0 S L1 (L) L15 (L) L2
L20 12 DUP REM L16 (23 DUPLICATES REMOVED)
L21 421 S L6
L22 222 S L21 (S) L1
L23 222 S L21 AND L22
L24 0 S L1 (S) L15 (S) L21
L25 0 S L1 (L) L15 (L) L21
L26 1 S L1 AND L15 AND L21
L27 99 DUP REM L22 (123 DUPLICATES REMOVED)
L28 21 S L1 AND L11
L29 8 S L27 AND L28
L30 8 DUP REM L29 (0 DUPLICATES REMOVED)

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-4.96	-4.96

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SESSION RESUMED IN FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE'
AT 15:16:44 ON 18 JUL 2002
FILE 'CAPLUS' ENTERED AT 15:16:44 ON 18 JUL 2002
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)        SINCE FILE      TOTAL
                                                     ENTRY     SESSION
CA SUBSCRIBER PRICE                                  -4.96        -4.96

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     (FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

     FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002
L1         19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
L2           509 S PROLYL ?HYDROXYLASE
L3             2 S L1 (S) L2
L4             2 S L1 (L) L2
L5             7 S DPY-18
L6           103 S DPY-18 OR DPY
L7            59 S L1 (L) L6
L8             0 S L7 AND L2

     FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002
L9            11 S L3
L10            6 DUP REM L9 (5 DUPLICATES REMOVED)
L11           25 S L5
L12            0 S EMBRYONAL LETHAL PHENOTYPE
L13          283 S EMBRYONIC LETHAL PHENOTYPE
L14          290 S EMBRYON? LETHAL PHENOTYPE
L15          290 S EMBRYON? LETHA? PHENOTYPE
L16           35 S L1 (S) L15
L17            0 S L16 AND L2
L18            0 S L1 (S) L15  (S) L2
L19            0 S L1 (L) L15  (L) L2
L20           12 DUP REM L16 (23 DUPLICATES REMOVED)
L21          421 S L6
L22          222 S L21 (S) L1
L23          222 S L21 AND L22
L24            0 S L1 (S) L15 (S) L21
L25            0 S L1 (L) L15 (L) L21
L26            1 S L1 AND L15 AND L21
L27           99 DUP REM L22 (123 DUPLICATES REMOVED)
L28           21 S L1 AND L11
L29            8 S L27 AND L28
L30            8 DUP REM L29 (0 DUPLICATES REMOVED)

=> D HIS

     (FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

     FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002
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     FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002
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L20           12 DUP REM L16 (23 DUPLICATES REMOVED)
L21          421 S L6
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L23          222 S L21 AND L22
L24            0 S L1 (S) L15 (S) L21
L25            0 S L1 (L) L15 (L) L21
L26            1 S L1 AND L15 AND L21
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photomorphogenesis and dwarfism (cpd) mutant. Measurements of endogenous brassinosteroid levels by gas chromatog.-mass spectrometry were consistent with this hypothesis. To examine brassinosteroid-regulated gene expression in dpy, we performed cDNA subtractive hybridization and isolated a novel xyloglucan endotransglycosylase that is regulated by brassinosteroid treatment. The curl-3 (cu-3) mutant (Lycopersicon pimpinellifolium [Jusl.] Mill.) shows extreme dwarfism, altered leaf morphol., de-etiolation, and reduced fertility, all strikingly similar to the Arabidopsis mutant brassinosteroid insensitive 1 (bri1). Primary root elongation of wild-type L. pimpinellifolium seedlings was strongly inhibited by brassinosteroid application, while cu-3 mutant roots were able to elongate at the same brassinosteroid concn. Moreover, cu-3 mutants retained sensitivity to indole-3-acetic acid, cytokinins, gibberellin, and abscisic acid while showing hypersensitivity to 2,4-dichlorophenoxyacetic acid in the root elongation assay. The cu-3 root response to hormones, coupled with its bri1-like phenotype, suggests that cu-3 may also be brassinosteroid insensitive.

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3

ACCESSION NUMBER: 1994:209702 CAPLUS
DOCUMENT NUMBER: 120:209702
TITLE: Molecular and genetic analyses of the Caenorhabditis elegans dpy-2 and dpy-10 collagen genes: A variety of molecular alterations affect organismal morphology
AUTHOR(S): Levy, Adam D.; Yang, Jie; Kramer, James M.
CORPORATE SOURCE: Med. Sch., Northwestern Univ., Chicago, IL, 60611, USA
SOURCE: Mol. Biol. Cell (1993), 4(8), 803-17
CODEN: MBCEEV; ISSN: 1059-1524
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The authors have identified and cloned the Caenorhabditis elegans dpy-2 and dpy-10 genes and detd. that they encode collagens. Genetic data suggested that these genes are important in morphogenesis and possibly other developmental events. These data include the morphol. phenotypes exhibited by mutants, unusual genetic interactions with the sqt-1 collagen gene, and suppression of mutations in the glp-1 and mup-1 genes. The proximity of the dpy-2 and dpy-10 genes (3.5 kilobase) and the structural similarity of their encoded proteins (41% amino acid identity) indicate that dpy-2 and dpy-10 are the result of a gene duplication event. The genes do not, however, appear to be functionally redundant, because a dpy-10 null mutant is not rescued by the dpy-2 gene. In addn., full complementation between dpy-2 and dpy-10 can be demonstrated with all recessive alleles tested in trans. Sequence anal. of several mutant alleles of each gene was performed to det. the nature of the mol. defects that can cause the morphol. phenotypes. Glycine substitutions within the Gly-X-Y portion of the collagens can result in dumpy (Dpy), dumpy, left roller (DLRol), or temp.-sensitive DLRol phenotypes. Dpy-10(cn64), a dominant temp.-sensitive DLRol allele, creates an Arg-to-Cys substitution in the amino non-Gly-X-Y portion of the protein. Three dpy-10 alleles contain Tc1 insertions in the coding region of the gene. Dpy-10(cg36) (DLRol) creates a nonsense codon near the end of the Gly-X-Y region. The nature of this mutation, combined with genetic data, indicates that DLRol is the null phenotype of dpy-10. The **Dpy phenotype** results from reduced function of the dpy-10 collagen gene. The authors' results indicate that a variety of mol. defects in these collagens can result in severe morphol. changes in C. elegans.

the genetic results and suggesting that **C. elegans** can serve as a model system for the discovery of new inhibitors.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:324546 CAPLUS
DOCUMENT NUMBER: 133:86929
TITLE: Prolyl 4-hydroxylase is an essential procollagen-modifying enzyme required for exoskeleton formation and the maintenance of body shape in the **nematode** Caenorhabditis **elegans**
AUTHOR(S): Winter, Alan D.; Page, Antony P.
CORPORATE SOURCE: Wellcome Centre for Molecular Parasitology, Anderson College, The University of Glasgow, Glasgow, G11 6NU, UK
SOURCE: Molecular and Cellular Biology (2000), 20(11), 4084-4093
CODEN: MCEBD4; ISSN: 0270-7306
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The multienzyme complex prolyl 4-hydroxylase catalyzes the hydroxylation of proline residues and acts as a chaperone during collagen synthesis in multicellular organisms. The .beta. subunit of this complex is identical to protein disulfide isomerase (PDI). The free-living **nematode** **C. elegans** is encased in a collagenous exoskeleton and represents an excellent model for the study of collagen biosynthesis and extracellular matrix formation. In this study, we examd. prolyl 4-hydroxylase .alpha.-subunit (PHY; EC 1.14.11.2)- and .beta.-subunit (PDI; EC 5.3.4.1)-encoding genes with respect to their role in collagen modification and formation of the **C. elegans** exoskeleton. We identified genes encoding 2 PHYs and a single assocd. PDI and showed that all 3 are expressed in collagen-synthesizing ectodermal cells at times of maximal collagen synthesis. Disruption of the pdi gene via RNA interference resulted in embryonic lethality. Similarly, the combined phy genes are required for embryonic development. Interference with phy-1 resulted in a morphol. dumpy phenotype, which we detd. to be identical to the uncharacterized **dpy-18** locus. Two **dpy-18** mutant strains were shown to have null alleles for phy-1 and to have a reduced hydroxyproline content in their exoskeleton collagens. This study demonstrates in vivo that this enzyme complex plays a central role in extracellular matrix formation and is essential for normal metazoan development.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:550782 CAPLUS
DOCUMENT NUMBER: 134:247771
TITLE: **dpy-18** encodes an .alpha.-subunit of prolyl-4-hydroxylase in Caenorhabditis **elegans**
AUTHOR(S): Hill, Katherine L.; Harfe, Brian D.; Dobbins, Carey A.; L'Hernault, Steven W.
CORPORATE SOURCE: Program in Genetics and Molecular Biology, Graduate Division of Biological and Biomedical Sciences, Emory University, Atlanta, GA, 30322, USA
SOURCE: Genetics (2000), 155(3), 1139-1148
CODEN: GENTAE; ISSN: 0016-6731
PUBLISHER: Genetics Society of America
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Collagen is an extracellular matrix (ECM) component encoded by a large

multigene family in multicellular animals. Procollagen is post-translationally modified by prolyl-4-hydroxylase (EC 1.14.11.2) before secretion and participation in ECM formation. Therefore, collagen processing and regulation can be studied by examg. this required interaction of prolyl-4-hydroxylase with procollagen. High-resoln. polymorphism mapping was used to place the Caenorhabditis **elegans** **dpy-18** gene on the phys. map, and we show that it encodes a prolyl-4-hydroxylase .alpha. catalytic subunit. The Dpy phenotype of **dpy-18**(e364) amber mutants is more severe when this mutation is in trans to the noncomplementing deficiency tDf7, while the **dpy-18**(e499) deletion mutant exhibits the same phenotype as **dpy-18**(e499)/tDf7. Furthermore, **dpy-18** RNA interference (RNAi) in wild-type worms results in Dpy progeny, while **dpy-18** (RNAi) in **dpy-18**(e499) mutants does not alter the Dpy phenotype of their progeny. These observations suggest that the **dpy-18** null phenotype is Dpy. A **dpy-18**::gfp promoter fusion construct is expressed throughout the hypodermis within the cells that abundantly produce the cuticle collagens, as well as in certain head and posterior neurons. While prolyl-4-hydroxylase has been studied extensively by biochem. techniques, this is the first report of a mutationally defined prolyl-4-hydroxylase in any animal.

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L30 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:323602 BIOSIS
DOCUMENT NUMBER: PREV200000323602
TITLE: Mutations with sensory ray defect unmask cuticular glycoprotein antigens in Caenorhabditis **elegans** male tail.
AUTHOR(S): Ko, Frankie C. F.; Chow, King L. (1)
CORPORATE SOURCE: (1) Department of Biology, Hong Kong University of Science and Technology, Clear Water Bay, Kowloon, Hong Kong China
SOURCE: Development Growth & Differentiation, (Feb., 2000) Vol. 42, No. 1, pp. 69-77. print.
ISSN: 0012-1592.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Caenorhabditis **elegans** male tail has nine pairs of bilaterally symmetric ray processes extended into a cuticular fan. The formation of these structures involves both cell lineage differentiation and cellular morphogenesis. Nine mutations were examined, all of which presented an amorphous ray phenotype. Glycoconjugates carrying an N-acetylglucosamine (GlcNAc) epitope were detected at a high level in their male bursa. It was shown that these antigens are not responsible for the morphological defects. It was further demonstrated that these ram and mab gene products represent critical components for male tail cuticle organization. Mutations of them abolish the integrity of the male bursal cuticle and unmask the underlying GlcNAc epitope.

L30 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:116067 CAPLUS
DOCUMENT NUMBER: 114:116067
TITLE: Properties of a class of genes required for ray morphogenesis in Caenorhabditis **elegans**
AUTHOR(S): Baird, Scott E.; Emmons, Scott W.
CORPORATE SOURCE: Dep. Mol. Genet., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SOURCE: Genetics (1990), 126(2), 335-44
CODEN: GENTAE; ISSN: 0016-6731
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Eight mutations were identified in Caenorhabditis **elegans** that define at least 5 terminal differentiation genes (ram genes) whose products are required during the extension of the male-specific ray sensilla. ram Gene mutations result in morphol. abnormalities in the sensory rays but do not appear to interfere with ray functions. A similar ray morphol. phenotype was obsd. in males harboring mutations in 3 previously defined genes, dpy-11, **dpy-18**, and sqt-1, that also affect body shape. One of these genes, sqt-1, is known to encode a collagen. Mutations in different ram genes failed to complement, suggesting that their gene products functionally interact. For one ram gene, failure to complement was shown to result from haploinsufficiency. Intergenic noncomplementation did not extend to the body morphol. genes. The temp.-sensitive periods of both ram and body morphol. mutations corresponded to the period of development in which ray extension occurs. It is proposed that ram gene products act together in a crit. interaction between the rays and the cuticle required for wild-type ray morphol.

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L28 21 S L1 AND L11
L29 8 S L27 AND L28
L30 8 DUP REM L29 (0 DUPLICATES REMOVED)

=> LOGOFF HOLD

L26 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:454514 CAPLUS
DOCUMENT NUMBER: 129:212270
TITLE: Isolation and characterization of lethal mutation near the unc-29 (LG I) region of Caenorhabditis **elegans**
AUTHOR(S): Lee, Jinsook; Ahnn, Joohong
CORPORATE SOURCE: Department of Life Science, Kwangju Institute of Science and Technology, Kwangju, 506-712, S. Korea
SOURCE: Korean Journal of Biological Sciences (1998), 2(1), 123-131
CODEN: KJBSFZ; ISSN: 1226-5071
PUBLISHER: Korean Association of Biological Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The unc-29 region on the chromosome I of Caenorhabditis **elegans** has been mutagenized in order to obtain lethal mutations. In this screen, the uncoordinated phenotype of unc-29 (e193) mutant was used to identify any lethal mutations closely linked to the unc-29 gene, which encodes a subunit of nicotinic acetylcholine receptors. The authors have isolated six independent mutations (jh1 to jh6) out of approx. 5,200 Et methanesulfonate (EMS) treated haploids. Four of the six mutations demonstrated **embryonic lethal phenotypes**, while the other two showed embryonic and larval lethal phenotypes. Terminal phenotypes obsd. in two mutations (jh1 and jh2) indicated developmental defects specific to posterior part of embryos which appeared similar to the phenotypes obsd. in nob (no back end) mutants. Another mutation (jh4) resulted in an interesting phenotype of body-wall muscle degeneration at larval stage. These mutations were mapped by using three-factor crosses and deficiency mutants in this region. Here the authors report genetic anal. and characterization of these lethal mutations.

regulation of MLS-1 at several different levels. The mls-1 promoter is activated by the **C. elegans** orthologs of Twist and Daughterless, but is only active in a subset of the lineage where these two transcription factors are present. mls-1 activity also appears to be regulated by posttranscriptional processes, as expression occurs in both uterine and vulval muscle precursors.

L30 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:68425 BIOSIS
DOCUMENT NUMBER: PREV200200068425
TITLE: Role of **C. elegans** lin-40 MTA in vulval fate specification and morphogenesis.
AUTHOR(S): Chen, Zhe; Han, Min (1)
CORPORATE SOURCE: (1) Department of Molecular, Cellular and Developmental Biology, Howard Hughes Medical Institute, University of Colorado at Boulder, Boulder, CO, 80309: mhan@colorado.edu USA
SOURCE: Development (Cambridge), (December, 2001) Vol. 128, No. 23, pp. 4911-4921. http://dev.biologists.org/current.shtml. print.
ISSN: 0950-1991.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Vulval differentiation in Caenorhabditis **elegans** involves several fundamental cellular events, including cell fusion, division and migration. We have characterized the role of the lin-40 (also known as egr-1) gene in these cellular processes. LIN-40 is homologous to the metastasis-associated factor 1 (MTA1) in mammals, which has been identified as a component of the nucleosome remodeling and histone deacetylation (NuRD) complex that functions as a transcriptional co-repressor. We show here that lin-40 negatively regulates vulval fate specification at least partly by promoting cell fusion between the vulval precursor cells and the hypodermal syncytium at an early larval stage. This inhibitory function of lin-40 might be carried out by downregulating lin-39 Hox expression. We also show that lin-40 is specifically required for cell divisions along the transverse orientation during vulval morphogenesis.

L30 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:296070 CAPLUS
DOCUMENT NUMBER: 133:233493
TITLE: Prolyl 4-hydroxylase is required for viability and morphogenesis in Caenorhabditis **elegans**
AUTHOR(S): Friedman, Lisa; Higgin, Joshua J.; Moulder, Gary; Barstead, Robert; Raines, Ronald T.; Kimble, Judith
CORPORATE SOURCE: Department of Biochemistry, University of Wisconsin, Madison, WI, 53706, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2000), 97(9), 4736-4741
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The genome of Caenorhabditis **elegans** possesses two genes, **dpy-18** and phy-2, that encode .alpha. subunits of the enzyme prolyl 4-hydroxylase. Th authors have generated deletions within each gene to eliminate prolyl 4-hydroxylase activity from the animal. The **dpy-18** mutant has an aberrant body morphol., consistent with a role of prolyl 4-hydroxylase in formation of the body cuticle. The phy-2 mutant is phenotypically wild type. However, the **dpy-18**; phy-2 double mutant is not viable, suggesting an essential role for prolyl 4-hydroxylase that is normally accomplished by either **dpy-18** or phy-2. The effects of the double mutation were mimicked by small-mol. inhibitors of prolyl 4-hydroxylase, validating

L30 ANSWER 1 OF 8 MEDLINE
ACCESSION NUMBER: 2002354365 IN-PROCESS
DOCUMENT NUMBER: 22092148 PubMed ID: 12097347
TITLE: High-Throughput Gene Mapping in Caenorhabditis **elegans**.
AUTHOR: Swan Kathryn A; Curtis Damian E; McKusick Kathleen B; Voinov Alexander V; Mapa Felipa A; Cancilla Michael R
CORPORATE SOURCE: Exelixis, Inc., South San Francisco, California 94083-0511, USA.
SOURCE: GENOME RESEARCH, (2002 Jul) 12 (7) 1100-5. Journal code: 9518021. ISSN: 1088-9051.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20020707
Last Updated on STN: 20020707

AB Positional cloning of mutations in model genetic systems is a powerful method for the identification of targets of medical and agricultural importance. To facilitate the high-throughput mapping of mutations in Caenorhabditis **elegans**, we have identified a further 9602 putative new single nucleotide polymorphisms (SNPs) between two **C**. **elegans** strains, Bristol N2 and the Hawaiian mapping strain CB4856, by sequencing inserts from a CB4856 genomic DNA library and using an informatics pipeline to compare sequences with the canonical N2 genomic sequence. When combined with data from other laboratories, our marker set of 17,189 SNPs provides even coverage of the complete worm genome. To date, we have confirmed >1099 evenly spaced SNPs (one every 91 +/- 56 kb) across the six chromosomes and validated the utility of our SNP marker set and new fluorescence polarization-based genotyping methods for systematic and high-throughput identification of genes in **C**. **elegans** by cloning several proprietary genes. We illustrate our approach by recombination mapping and confirmation of the mutation in the cloned gene, **dpy-18**. [The sequence data described in this paper have been submitted to the NCBI dbSNP data library under accession nos. 4388625-4389689 and GenBank dbSTS under accession nos. 973810-974874. The following individuals and institutions kindly provided reagents, samples, or unpublished information as indicated in the paper: The **C**. **elegans** Sequencing Consortium and The Caenorhabditis Genetics Center.]

L30 ANSWER 2 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:141383 BIOSIS
DOCUMENT NUMBER: PREV200200141383
TITLE: The T-box factor MLS-1 acts as a molecular switch during specification of nonstriated muscle in **C**. **elegans**.
AUTHOR(S): Kostas, Stephen A.; Fire, Andrew (1)
CORPORATE SOURCE: (1) Department of Embryology, Carnegie Institution of Washington, Baltimore, MD, 21210: fire@ciwemb.edu USA
SOURCE: Genes & Development, (January 15, 2002) Vol. 16, No. 2, pp. 257-269. http://www.genesdev.org/. print. ISSN: 0890-9369.
DOCUMENT TYPE: Article
LANGUAGE: English

AB We have isolated mutations in a gene mls-1 that is required for proper specification of nonstriated muscle fates in Caenorhabditis **elegans**. Loss of MLS-1 activity causes uterine muscle precursors to forego their normal fates, instead differentiating as vulval muscles. We have cloned mls-1 and shown that the product is a member of the T-box family of transcriptional regulators. MLS-1 acts as a cell fate determinant in that ectopic expression can transform other cell types to uterine muscle precursors. Uterine muscle patterning is executed by

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 NEWS  3  Jan 29  FSTA has been reloaded and moves to weekly updates
 NEWS  4  Feb 01  DKILIT now produced by FIZ Karlsruhe and has a new update
                  frequency
 NEWS  5  Feb 19  Access via Tymnet and SprintNet Eliminated Effective 3/31/02
 NEWS  6  Mar 08  Gene Names now available in BIOSIS
 NEWS  7  Mar 22  TOXLIT no longer available
 NEWS  8  Mar 22  TRCTHERMO no longer available
 NEWS  9  Mar 28  US Provisional Priorities searched with P in CA/CAplus
                  and USPATFULL
 NEWS 10  Mar 28  LIPINSKI/CALC added for property searching in REGISTRY
 NEWS 11  Apr 02  PAPERCHEM no longer available on STN.  Use PAPERCHEM2 instead.
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 NEWS 17  Apr 22  BIOSIS Gene Names now available in TOXCENTER
 NEWS 18  Apr 22  Federal Research in Progress (FEDRIP) now available
 NEWS 19  Jun 03  New e-mail delivery for search results now available
 NEWS 20  Jun 10  MEDLINE Reload
 NEWS 21  Jun 10  PCTFULL has been reloaded
 NEWS 22  Jul 02  FOREGE no longer contains STANDARDS file segment

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               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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For a list of commands available to you in the current file, enter
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=> FILE MEDLINE
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                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 0.21       0.21
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=> S NEMATODE OR C.ELEGANS OR ?ELEGANS
         11672 NEMATODE
          4439 NEMATODES
         13874 NEMATODE
                 (NEMATODE OR NEMATODES)
        728697 C
          7442 ELEGANS
          2724 C.ELEGANS
                 (C(W)ELEGANS)
          7443 ?ELEGANS
L1       19214 NEMATODE OR C.ELEGANS OR ?ELEGANS

=> S PROLYL?HYDROXYLASE
'?' TRUNCATION SYMBOL NOT VALID WITHIN 'PROLYL?HYDROXYLASE'
The truncation symbol ?  may be used only at the end of a search
term.  To specify a variable character within a word use '!', e.g.,
'wom!n' to search for both 'woman' and 'women'.  Enter "HELP
TRUNCATION" at an arrow prompt (=>) for more information.

=> S PROLYL ?HYDROXYLASE
          4465 PROLYL
         30651 ?HYDROXYLASE
L2         509 PROLYL ?HYDROXYLASE
                 (PROLYL(W)?HYDROXYLASE)

=> S L1 (s) L2
L3           2 L1 (S) L2

=> S L1 (L) L2
L4           2 L1 (L) L2

=> D 1-2 IBIB ABS

L4   ANSWER 1 OF 2     MEDLINE
ACCESSION NUMBER:   2001548313     MEDLINE
DOCUMENT NUMBER:    21479120   PubMed ID: 11595184
TITLE:              C. elegans EGL-9 and mammalian homologs define a family of
                    dioxygenases that regulate HIF by prolyl hydroxylation.
COMMENT:            Comment in: Cell. 2001 Oct 5;107(1):1-3
AUTHOR:             Epstein A C; Gleadle J M; McNeill L A; Hewitson K S;
                    O'Rourke J; Mole D R; Mukherji M; Metzen E; Wilson M I;
                    Dhanda A; Tian Y M; Masson N; Hamilton D L; Jaakkola P;
                    Barstead R; Hodgkin J; Maxwell P H; Pugh C W; Schofield C
                    J; Ratcliffe P J
CORPORATE SOURCE:   The Henry Wellcome Building of Genomic Medicine, Roosevelt
                    Drive, Oxford OX3 7BN, United Kingdom.
SOURCE:             CELL, (2001 Oct 5) 107 (1) 43-54.
                    Journal code: 0413066. ISSN: 0092-8674.
PUB. COUNTRY:       United States
                    Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200112
ENTRY DATE:         Entered STN: 20011015
                    Last Updated on STN: 20020420
                    Entered Medline: 20011204
AB   HIF is a transcriptional complex that plays a central role in mammalian
     oxygen homeostasis. Recent studies have defined posttranslational
     modification by prolyl hydroxylation as a key regulatory event that
     targets HIF-alpha subunits for proteasomal destruction via the von
     Hippel-Lindau ubiquitylation complex. Here, we define a conserved HIF-VHL-
       ***prolyl***     ***hydroxylase***   pathway in   ***C***   .
       ***elegans***   , and use a genetic approach to identify EGL-9 as a
     dioxygenase that regulates HIF by prolyl hydroxylation. In mammalian
```

series of isoforms bearing a conserved 2-histidine-1-carboxylate iron coordination motif at the catalytic site. Direct modulation of recombinant enzyme activity by graded hypoxia, iron chelation, and cobaltous ions mirrors the characteristics of HIF induction in vivo, fulfilling requirements for these enzymes being oxygen sensors that regulate HIF.

L4 ANSWER 2 OF 2 MEDLINE
ACCESSION NUMBER: 79021663 MEDLINE
DOCUMENT NUMBER: 79021663 PubMed ID: 212107
TITLE: In vitro translation of nematode cuticular collagens.
AUTHOR: Noble S; Leushner J; Pasternak J
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1978 Aug 23) 520 (1) 219-28.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197812
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19781220
AB Phenanthroline treatment of growing cultures of the free-living ***nematode*** Panagrellus silusiae was used to lower the degree of hydroxylation of nascent collagen chains at the polysomal level. Under these conditions, the bound pentasome-hexasome fraction provided substrate for ***prolyl*** ***hydroxylase*** . When this polysomal fraction was subsequently tested in a cell-free wheat germ system, collagenase-susceptible translation products were observed after sodium dodecyl sulfate-acrylamide gel electrophoresis. The electrophoretic mobilities of each of these four major collagen products were similar to four collagens that are isolated from intact cuticles. In addition, purified polysomal RNA that adhered to unmodified cellulose directed the synthesis of four pepsin-resistant polypeptides that had molecular weights that coincided with four pepsin-resistant collagens that can be purified from the cuticle of this species. Thus, the polysomal site of the messenger RNAs for the cuticular collagens of P. silusiae was located. Although precursor forms of the cuticular collagens were not produced in the cell-free system, the question whether additional amino acid segments occur on the primary translational products of the cuticular collagens in vivo remains open.

=> D HIS

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FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002
L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
L2 509 S PROLYL ?HYDROXYLASE
L3 2 S L1 (S) L2
L4 2 S L1 (L) L2

=> S DPY-18
103 DPY
365958 18
L5 7 DPY-18
(DPY(W)18)

=> S DPY-18 OR DPY
103 DPY
365958 18
7 DPY-18
(DPY(W)18)
103 DPY
L6 103 DPY-18 OR DPY

=> S L1 (L) L6
L7 59 L1 (L) L6

=> S L7 AND L2
L8 0 L7 AND L2

=> D HIS

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